Impaired Volitional Closure of the Left Eyelid After Right Anterior Cerebral Artery Infarction

Apraxia Due to Interhemispheric Disconnection?

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Background: The inability of volitional unilateral eyelid closure is an uncommon symptom of a central nervous system disorder. When it occurs as the result of a localized brain lesion, it is debated to be a form of supranuclear facial palsy or an apraxic phenomenon.

Objectives: To report and discuss a unilateral (left-sided) higher-order movement disorder of the facial periorcular musculature bearing apraxic features.

Setting: University neurology department.

Patient: A 78-year-old right-handed man was admitted to the hospital with a left-sided brachiofacial hemiparesis of sudden onset. After thrombolysis with intravenous recombinant tissue-type plasminogen activator, the hemiparesis, including the left-sided facial weakness, disappeared. Serial computed tomographic scans showed that the patient was left with a stroke in the right anterior cerebral artery territory, affecting the frontal commissural fibers of the corpus callosum. There were no signs of upper motor neuron facial paresis on the left side when gesturing in a natural context. Eyelid closure was complete during sleep. However, left eyelid closure and elevation of the left eyebrow were not possible on verbal command. In contrast, voluntary innervation of the perioral facial musculature was performed properly.

Conclusions: The voluntary-automatic dissociation of our patient’s eyelid closure was suggestive of an apraxic disorder. Disconnection from a praxis center caused by callosal damage may be assumed to be the underlying cause. The unilaterality of the symptom might imply that in a bilaterally organized corticonuclear system such as upper face innervation, it is the crossing fibers that are primarily involved in praxis tasks.

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left arm. Eight years previously, he had to be treated with bypass surgery because of coronary heart disease. Four
years before, a pacemaker was implanted because of atrio-
ventricular blockage. His main cardiovascular risk fac-
tor was arterial hypertension.

At examination, the patient was alert and fully ori-
tented, with fluent speech. He had a conjugated devia-
tion of gaze to the right but no gaze palsy to the left. A
medium-grade paresis of the left-sided lower facial muscles
was evident. The other cranial nerve functions were pre-
served. The left-sided hemiparesis was high grade in the
arm and minor in the leg. The deep tendon reflexes of
the left arm were increased. The left plantar response was
extensor. The initial computed tomographic scan did not
show signs of an ischemic stroke but excluded hemor-
rhage. Magnetic resonance imaging was not possible since
the patient had a pacemaker.

A systemic thrombolysis with intravenous recom-
binant tissue-type plasminogen activator (rtPA) (1 mg/
kg) was performed. One day later, the patient exhibited
normal strength in his left arm and hand. However, it
turned out to be very difficult for him to voluntarily close
his left eye. While he could easily puff up his cheeks and
hold in the air, it was virtually impossible for him to close
his left eye on verbal command (Figure, A and B). He
was unable to lift his left eyebrow when told to do so nor
could he keep it elevated when it had been lifted pass-
vively by the examiner. In contrast, the involuntary eye-
lid closure during sleep was not affected and neither was
the blink reflex. There was no impairment of automatic
gesticulation (smiling, laughing, or blinking) outside the
clinical test setting.

Control computed tomographic scans were per-
formed 11 hours and 10 days after thrombolysis. They
revealed an ischemic stroke in the right anterior cere-
bral artery territory (Figure, C). In the digital subtrace-
tion arteriogram, the proximal internal carotid artery on
the right side was found to be occluded and had not be-
come recanalized after the thrombolysis procedure,
whereas the distal part of the internal carotid artery and
the medial cerebral artery but not the anterior cerebral
artery on the right side were refilled via the ophthalmic
artery. Regarding the initial medial cerebral artery syn-
drome (left-sided brachiofacial hemiparesis), we there-
fore hypothesize that initially, the carotid bifurcation could
have been blocked by a thrombus whose medial cere-
bral artery “limb” might have been lysed successfully by
rtPA, leaving part of the anterior cerebral artery terri-
tory infarcted.

The impossibility of voluntary unilateral eyelid closure
due to a localized brain lesion was observed in our pa-
tient. Previous reports about the bilateral lack of voli-
tional eyelid closure as a pseudobulbar phenomenon
resulting from generalized or disseminated brain lesions
are abundant.2 The number of cases is getting rarer when
a localized lesion—especially in the right hemi-
sphere—is supposed to be the underlying cause.6,8 Ghika
et al5 considered cortical damage and loss of association
fibers between supplementary motor regions and the pre-
motor or primary motor areas responsible for the bilat-
eral inability of 2 patients with ischemic lesions involv-
ing Brodmann areas 4 and 6 of the right parietal lobe to
close their eyes voluntarily. Disconnection between these
areas would lead to failure of volitional eyelid closure.
The failure would be bilateral since eyelid closure is sup-
posed to be an axial movement with bilateral innerva-
tion. A systematic investigation of patients with lateral-
ized brain lesions for (bilateral) face apraxia has recently
been conducted, illustrating that 46% of left-hemisphere-
damaged and 44% of right-hemisphere-damaged pa-
tients showed apraxic upper face movements.9

Here we describe a patient with a disorder of vol-
untary movement in the upper portion of the left face af-
ter right anterior cerebral artery stroke. The intact sponta-
aneous facial gesturing revealed no evidence for left facial
weakness due to upper motor neuron damage. Also, a
supranuclear palsy of cranial nerve VII could hardly be
present on theoretical grounds since it is commonly true
for the innervation of the periorcular and forehead muscu-
larucity to be spared in contralateral stroke due to un-
crossed innervation of the corresponding part of the fa-
cial nucleus. If ever there is some variability in the
organization of the corticonuclear pathway to the facial
nucleus, it is in the way that the motoneurons of the lower
facial muscles (in addition to the upper ones) may receive bilateral corticonuclear input. Therefore, it is quite unlikely that the impaired voluntary movement of the left periocular muscles was due to right pyramidal (corticonuclear) tract damage.

Rather, we suggest that the lesion of the right limb of the frontal callosal forceps (Figure, C) led to disconnection of the right hemispheric motor system from the praxis center in the left hemisphere. The disconnection from the left intraparietal cortex (intraparietal sulcus) or the left dorsolateral frontal cortex is believed to be a substrate of ideomotor apraxia in right-handed subjects. The observed left-sided facial movement disorder was reminiscent of ideomotor apraxic features since there was a clear dissociation between impaired voluntary closure of the left eyelid on verbal command and perfectly normal blinking and automatic eyelid closure in a natural context. In our patient, the disturbance of voluntary gesturing was unilateral (confined to the left half of the face). Given the commonly accepted truth that the innervation of the upper face is bilateral with a contralateral predominance, we would like to hypothesize that the predominance of the crossing corticonuclear fibers is unmasked more clearly in volitionally evoked movements of the periocular musculature as compared with reflex movements.

Nevertheless, we cannot exclude that the described phenomenon is the facial equivalent of a limb-kineti
c apraxia (loss of deftness), which may result from right-sided damage to intrahemispheric premotor cortex connections, with subsequent alteration of motor engrams. However, a certain dominance of the left hemisphere has recently been shown. In contrast with the present case, a voluntary-automatic dissociation of motor performance is usually not observed in limb-kineti
c apraxia. In addition, according to the bilateral projections of the upper facial corticonuclear tract neurons, we would predict more prominent bilateral clinical signs if the intrahemispheric motor engram for the movement of the upper face was lesioned.

It has long been known that lesions of the callosal splenium can make individuals lose the very concept of skilled movements. This has been referred to as agnosia for tool usage and is associated with disconnection from the dominant posterior parietal lobe, whose projections cross over in the splenium. Isolated defects of the callosal trunk result in poor performance of a particular motor act in response to a verbal command (ideomotor apraxia). This type of apraxia is seen unilaterally in the left hand when the anterior callosal trunk is lesioned (“alien hand syndrome”).

As a conclusion from this case, the hypothesis might be raised that lesions involving the anterior parts of the callosal body could selectively cause left-sided dyspraxia that presents as isolated higher-order disturbance of facial movement. In this sort of “disconnection syndrome,” the preformed lateralization of the bilaterally organized corticonuclear tract for upper face innervation may be unmasked.

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